EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	10	Tani near2 Kousuke	US-PGPUB	OR	ON	2006/04/05 10:59
L3	1	EP3 same autoimmune	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:04
L4	7	EP3 same allerg$	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:06
L5	29	EP3.ti,ab.	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:15
L6	6	EP3.ti,ab. and (allerg$ or asthma)	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:07
L7	1619	"prostaglandin E2"	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:16
L8	849	"prostaglandin E2" and (allerg$ or asthma)	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:16
L9	49	"prostaglandin E2" same (allerg$ or asthma)	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:16
L10	10	"prostaglandin E2" with (allerg$ or asthma)	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:16
S1	2	"6642266".pn. "4386031".pn.	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 10:59
S2	2	EP3 with allerg$	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:04
S3	7	EP3 same allerg$	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:04
S4	211	strong.in.	US-PGPUB	OR	ON	2006/03/24 07:19
S5	1	strong.in. and cox-2.ti.	US-PGPUB	OR	ON	2006/03/24 07:29
S6	0	("2868691"|"3095355"|"3965143"|"3985791"|"4301146"|"4692464"|"4863961"|"5624959"|"5723493"|"5753700"|"6013673").PN.	US-PGPUB	OR	ON	2006/03/24 07:29
S7	11	("2868691"|"3095355"|"3965143"|"3985791"|"4301146"|"4692464"|"4863961"|"5624959"|"5723493"|"5753700"|"6013673").PN.	US-PGPUB; USPAT; EPO	OR	ON	2006/03/24 07:31

EAST Search History

S8	1	"5157052".pn.	US-PGPUB; USPAT; EPO	OR	ON	2006/03/24 07:31

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                    USPAT2
NEWS  5  JAN 13  IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
NEWS  6  JAN 13  New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to
                 INPADOC
NEWS  7  JAN 17  Pre-1988 INPI data added to MARPAT
NEWS  8  JAN 17  IPC 8 in the WPI family of databases including WPIFV
NEWS  9  JAN 30  Saved answer limit increased
NEWS 10  JAN 31  Monthly current-awareness alert (SDI) frequency
                 added to TULSA
NEWS 11  FEB 21  STN AnaVist, Version 1.1, lets you share your STN AnaVist
                 visualization results
NEWS 12  FEB 22  Status of current WO (PCT) information on STN
NEWS 13  FEB 22  The IPC thesaurus added to additional patent databases on STN
NEWS 14  FEB 22  Updates in EPFULL; IPC 8 enhancements added
NEWS 15  FEB 27  New STN AnaVist pricing effective March 1, 2006
NEWS 16  FEB 28  MEDLINE/LMEDLINE reload improves functionality
NEWS 17  FEB 28  TOXCENTER reloaded with enhancements
NEWS 18  FEB 28  REGISTRY/ZREGISTRY enhanced with more experimental spectral
                 property data
NEWS 19  MAR 01  INSPEC reloaded and enhanced
NEWS 20  MAR 03  Updates in PATDPA; addition of IPC 8 data without attributes
NEWS 21  MAR 08  X.25 communication option no longer available after June 2006
NEWS 22  MAR 22  EMBASE is now updated on a daily basis
NEWS 23  APR 03  New IPC 8 fields and IPC thesaurus added to PATDPAFULL
NEWS 24  APR 03  Bibliographic data updates resume; new IPC 8 fields and IPC
                 thesaurus added in PCTFULL
NEWS 25  APR 04  STN AnaVist $500 visualization usage credit offered

NEWS EXPRESS  FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,
              CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
              AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
              V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT
              http://download.cas.org/express/v8.0-Discover/

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FILE 'HOME' ENTERED AT 11:56:16 ON 05 APR 2006

=> file medline
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.42       0.42

FILE 'MEDLINE' ENTERED AT 11:57:37 ON 05 APR 2006

 FILE LAST UPDATED: 4 APR 2006 (20060404/UP).  FILE COVERS 1950 TO DATE.

 On December 11, 2005, the 2006 MeSH terms were loaded.

 The MEDLINE reload for 2006 is now (26 Feb.) available.  For details
 on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>).
 See also:
```

http://www.nlm.nih.gov/mesh/
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> s EP3 (L) (allergy or asthma)
          654 EP3
        42129 ALLERGY
        90571 ASTHMA
L1          7 EP3 (L) (ALLERGY OR ASTHMA)

=> d 1-7 bib abs
```

L1 ANSWER 1 OF 7 MEDLINE on STN
AN 2005516639 IN-PROCESS
DN PubMed ID: 16189372
TI Evaluation of selective prostaglandin E2 (PGE2) receptor agonists as therapeutic agents for the treatment of asthma.
AU Chung K F
CS National Heart and Lung Institute, Imperial College London, London SW3 6LY, UK.. f.chung@imperial.ac.uk
SO Science's STKE [electronic resource] : signal transduction knowledge environment, (2005 Sep 27) Vol. 2005, No. 303, pp. pe47. Electronic Publication: 2005-09-27.
Journal code: 100964423. E-ISSN: 1525-8882.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals
ED Entered STN: 20050929
Last Updated on STN: 20051215
AB Prostaglandin E2 (PGE2) released in asthmatic airways has bronchodilator properties and inhibits allergen-induced bronchoconstriction and release of inflammatory mediators. Although considered as a potential treatment for **asthma**, PGE2 also has some proinflammatory properties. PGE2 acts through four different receptor subtypes (EP1, EP2, **EP3**, and EP4) that may explain some of PGE2's diverse effects. In a mouse model of allergic inflammation in which the four receptors were individually deleted, only **EP3**(-/-) mice showed an enhancement of inflammation, whereas an **EP3** agonist was inhibitory, with PGE2 being inactive. Thus, **EP3** agonists may lead to a new approach for the treatment of **asthma.** However, other PGE2 receptor subtypes may also have beneficial effects, and a greater understanding of the signaling pathways of these receptor subtypes will help to clarify the role of these receptors in **asthma.**

L1 ANSWER 2 OF 7 MEDLINE on STN
AN 2005209441 MEDLINE
DN PubMed ID: 15806106
TI Suppression of allergic inflammation by the prostaglandin E receptor subtype EP3.
AU Kunikata Tomonori; Yamane Hana; Segi Eri; Matsuoka Toshiyuki; Sugimoto Yukihiko; Tanaka Satoshi; Tanaka Hiroyuki; Nagai Hiroichi; Ichikawa Atsushi; Narumiya Shuh
CS Department of Pharmacology and Faculty of Medicine and Graduate School of Pharmaceutical Sciences, Kyoto University, Kyoto 606-8501, Japan.
SO Nature immunology, (2005 May) Vol. 6, No. 5, pp. 524-31. Electronic

Publication: 2005-04-03.
Journal code: 100941354. ISSN: 1529-2908.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200505
ED Entered STN: 20050422
Last Updated on STN: 20050517
Entered Medline: 20050516
AB Prostaglandins, including PGD(2) and PGE(2), are produced during allergic reactions. Although PGD(2) is an important mediator of allergic responses, aspirin-like drugs that inhibit prostaglandin synthesis are generally ineffective in allergic disorders, suggesting that another prostaglandin-mediated pathway prevents the development of allergic reactions. Here we show that such a pathway may be mediated by PGE(2) acting at the prostaglandin E receptor **EP3**. Mice lacking **EP3** developed allergic inflammation that was much more pronounced than that in wild-type mice or mice deficient in other prostaglandin E receptor subtypes. Conversely, an **EP3**-selective agonist suppressed the inflammation. This suppression was effective when the agonist was administered 3 h after antigen challenge and was associated with inhibition of **allergy**-related gene expression. Thus, the PGE(2)-**EP3** pathway is an important negative modulator of allergic reactions.

L1 ANSWER 3 OF 7 MEDLINE on STN
AN 2004537201 MEDLINE
DN PubMed ID: 15257985
TI Augmentation of bovine airway smooth muscle responsiveness to carbachol, KCl, and histamine by the isoprostane 8-iso-PGE2.
AU Catalli Adriana; Janssen Luke J
CS Firestone Institute for Respiratory Health and Father Sean O'Sullivan Research Center, St. Joseph's Hospital, Hamilton, Ontario, Canada.
SO American journal of physiology. Lung cellular and molecular physiology, (2004 Nov) Vol. 287, No. 5, pp. L1035-41. Electronic Publication: 2004-07-16.
Journal code: 100901229. ISSN: 1040-0605.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200411
ED Entered STN: 20041029
Last Updated on STN: 20041219
Entered Medline: 20041119
AB Isoprostanes are generated during periods of oxidative stress, which characterize diseases such as **asthma** and cystic fibrosis. They also elicit functional responses and may therefore contribute to the pathology of these diseases. We set out to examine the effects of isoprostanes on airway responsiveness to cholinergic stimulation. Muscle bath techniques were employed using isolated bovine tracheal smooth muscle. 8-Isoprostaglandin E2 (8-iso-PGE2) increased tone directly on its own, although the magnitude of this response, even at the highest concentration tested, was only a fraction of that evoked by KCl or carbachol. More importantly, though, pretreatment of the tissues with 8-iso-PGE2 (10 microM) markedly augmented responses to submaximal and even subthreshold concentrations of KCl, carbachol, or histamine, whereas maximal responses to these agents were unaffected by the isoprostane. The augmentative effect on cholinergic responsiveness was mimicked by PGE2 (0.1 microM) and by the FP agonists PGF2 (0.1 microM) and fluprostenol (0.1 microM), but not by the **EP3** agonist sulprostone (0.1 microM) or the TP agonist U-46619 (0.1 microM). Antagonists of EP1 receptors (AH-6809 and SC-19920, 10 microM) and TP receptors (ICI-192605, 1 microM) had no effect on 8-iso-PGE2-induced augmentation of cholinergic

responsiveness. We conclude that 8-iso-PGE2 induces nonspecific airway smooth muscle hyperresponsiveness through a non-TP non-EP prostanoid receptor.

L1 ANSWER 4 OF 7 MEDLINE on STN
AN 2004231827 MEDLINE
DN PubMed ID: 15131569
TI Increased sensitivity of asthmatic airway smooth muscle cells to prostaglandin E2 might be mediated by increased numbers of E-prostanoid receptors.
AU Burgess Janette K; Ge Qi; Boustany Sarah; Black Judith L; Johnson Peter R A
CS Respiratory Research Group, Department of Pharmacology, Bosch Building D05, University of Sydney, Sydney, NSW 2006, Australia.
SO The Journal of allergy and clinical immunology, (2004 May) Vol. 113, No. 5, pp. 876-81.
Journal code: 1275002. ISSN: 0091-6749.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200406
ED Entered STN: 20040510
Last Updated on STN: 20040618
Entered Medline: 20040617
AB BACKGROUND: An increase in airway smooth muscle (ASM) cell proliferation leads to an increase in the bulk of the ASM, one of the characteristic features of **asthma**. We have previously shown that ASM cells from asthmatic individuals proliferate more than those from nonasthmatic subjects. This increased growth might be due to compromised inhibitory mechanisms within the ASM of asthmatic subjects. OBJECTIVE: The purpose of this study was to determine whether the proliferative control exerted by prostaglandin E(2) (PGE(2)) was altered in the asthmatic ASM cells. METHODS: We used tritated thymidine uptake to measure cell proliferation and cell-surface ELISAs to detect the presence of cell-surface receptors on ASM cells isolated from asthmatic and nonasthmatic individuals. RESULTS: The asthmatic ASM cells were significantly more sensitive to proliferation inhibition by PGE(2) than the nonasthmatic cells (P<.02). The PGE(2) (E-prostanoid [EP]) receptors EP2 and **EP3** were detected on asthmatic and nonasthmatic smooth muscle cells in culture. There were significantly more receptors on the asthmatic cells. The asthmatic cells also had increased sensitivity to proliferation inhibition by EP2-specific agonists but not by **EP3**-specific agonists. CONCLUSION: The increased growth observed in asthmatic ASM cells is not the result of impaired responsiveness to PGE(2). In contrast, these cells have increased sensitivity. This increased sensitivity might be mediated by the increased numbers of EP2 receptors on the surface.

L1 ANSWER 5 OF 7 MEDLINE on STN
AN 2003137787 MEDLINE
DN PubMed ID: 12618422
TI Receptors and pathways mediating the effects of prostaglandin E2 on airway tone.
AU Tilley Stephen L; Hartney John M; Erikson Christopher J; Jania Corey; Nguyen Mytrang; Stock Jeffrey; McNeisch John; Valancius Cathy; Panettieri Reynold A Jr; Penn Raymond B; Koller Beverly H
CS Department of Medicine, Division of Pulmonary and Critical Care Medicine, University of North Carolina at Chapel Hill, 27599-7248, USA.
NC HL-04280 (NHLBI)
HL-58506 (NHLBI)
HL-68141 (NHLBI)
SO American journal of physiology. Lung cellular and molecular physiology, (2003 Apr) Vol. 284, No. 4, pp. L599-606. Electronic Publication: 2002-12-13.
Journal code: 100901229. ISSN: 1040-0605.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200304
ED Entered STN: 20030326
Last Updated on STN: 20030416
Entered Medline: 20030410
AB Prostaglandin E(2) (PGE(2)) has complex effects on airway tone, and the existence of four PGE(2) [E-prostanoid (EP)] receptors, each with distinct signaling characteristics, has provided a possible explanation for the seemingly contradictory actions of this lipid mediator. To identify the receptors mediating the actions of PGE(2) on bronchomotor tone, we examined its effects on the airways of wild-type and EP receptor-deficient mice. In conscious mice the administration of PGE(2) increased airway responsiveness primarily through the EP1 receptor, although on certain genetic backgrounds a contribution of the **EP3** receptor was detected. These effects of PGE(2) were eliminated by pretreatment with either atropine or bupivacaine and were undetectable in anesthetized mice or in denervated tracheal rings, where only EP2-mediated relaxation of airway smooth muscle was observed. Together, our findings are consistent with a model in which PGE(2) modulates airway tone by activating multiple receptors expressed on various cell populations and in which the relative contribution of these receptors might depend on the expression of modifier alleles. PGE(2)/EP1/**EP3**-induced airway constriction occurs indirectly through activation of neural pathways, whereas PGE(2)-induced bronchodilation results from direct activation of EP2 receptors on airway smooth muscle. This segregation of EP receptor function within the airway suggests that PGE(2) analogs that selectively activate the EP2 receptor without activating the EP1/**EP3** receptors might prove useful in the treatment of **asthma.**

L1 ANSWER 6 OF 7 MEDLINE on STN
AN 2002242595 MEDLINE
DN PubMed ID: 11979724
TI The roles of the prostanoids played in the body.
AU Ushikubi Fumitaka; Narumiya Shuh
CS Department of Pharmacology, Asahikawa Medical College, Midorigaoka-Higashi 2-1-1-1, Asahikawa 078-8510, Japan.
SO Nippon yakurigaku zasshi. Folia pharmacologica Japonica, (2002 Apr) Vol. 119, No. 4, pp. 201-7. Ref: 13
Journal code: 0420550. ISSN: 0015-5691.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA Japanese
FS Priority Journals
EM 200206
ED Entered STN: 20020501
Last Updated on STN: 20020619
Entered Medline: 20020618
AB The actions of prostanoids in various physiological and pathophysiological conditions have been examined using mice lacking the prostanoid receptors. PGD2 was found to be a mediator of allergic **asthma.** Prostaglandin (PG) I2 worked not only as a mediator of inflammation but also as an antithrombotic and cardio-protective agent. Several important actions of PGE2 are brought out via the PGE2-receptor subtype **EP3** ; PGE2 participated in the regulation of platelet function, and it worked as a mediator of febrile responses to both endogenous and exogenous pyrogens. These novel findings on the roles of the prostanoids would contribute to the development of drugs targeting the prostanoid receptors.

L1 ANSWER 7 OF 7 MEDLINE on STN
AN 2000448157 MEDLINE
DN PubMed ID: 11001172

TI Roles of prostanoids revealed from studies using mice lacking specific prostanoid receptors.
AU Ushikubi F; Sugimoto Y; Ichikawa A; Narumiya S
CS Department of Pharmacology, Asahikawa Medical College, Japan.
SO Japanese journal of pharmacology, (2000 Aug) Vol. 83, No. 4, pp. 279-85.
Ref: 61
Journal code: 2983305R. ISSN: 0021-5198.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 200102
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010202
AB The actions of prostanoids in various physiological and pathophysiological conditions have been being examined using mice lacking different prostanoid receptors. Prostaglandin (PG) I2 worked not only as a mediator of inflammation but also as an antithrombotic agent. PGF2alpha was found to be an essential inducer of labor. Several important actions of PGE2 are exerted via each of the four PGE2 receptor subtypes: EP1, EP2, **EP3** and EP4. PGE2 participated in colon carcinogenesis via the EP1. PGE2 also participates in ovulation and fertilization and contributes to the control of blood pressure under high-salt intake via the EP2. PGE2 worked as a mediator of febrile responses to both endogenous and exogenous pyrogens and as a regulator of bicarbonate secretion induced by acid-stimulation in the duodenum via the **EP3** . It regulated the closure of ductus arteriosus and showed bone resorbing action via the EP4. PGD2 was found to be a mediator of allergic **asthma.** These studies have revealed important roles of prostanoids, some of which had not previously been known.

=>

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STN INTERNATIONAL LOGOFF AT 12:06:12 ON 05 APR 2006